many false-positive abnormalities is inherent to the statistical assumptions of normalcy for each test and has no actual clinical benefit. Repeat confirmatory and further diagnostic tests to pursue such abnormal laboratory values also contribute to the costs of such screening. Preoperative laboratory testing has been projected in one study to cost about $4.2 million for each life lost. In an era of increasing cost-containment, such funds could be spent more efficiently to save more lives.

Similarly, the usefulness of more expensive preoperative tests, such as the electrocardiogram and chest x-ray film, to detect asymptomatic illnesses capable of affecting the surgical outcome has proved to be minimal in clinical studies of young, otherwise healthy patients. Unnecessary delays in a surgical procedure and the concomitant patient anxiety for the workup of benign diseases (for example, the asymptomatic granuloma presenting as a lung nodule) also contribute to the inadequacies of these tests. In the elderly in whom the number of diseases and their prevalence increase, chest radiographs and electrocardiograms may be more helpful.

Even when such preoperative laboratory tests are ordered, physicians fail more than half the time to note the abnormalities in the chart and to order follow-up tests.

These studies provide evidence that, in the absence of specific clinical indications, "routine" preoperative laboratory testing can be safely eliminated. Because it has been estimated that 50% of all surgical procedures in this country are done on presumably healthy persons, the potential cost savings would be enormous. It is reassuring to know that the "best test" preoperatively in minimizing surgical morbidity and mortality is still a careful history and physical examination.

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Weight Control in Managing Hypertension

NUMEROUS EPIDEMIOLOGIC STUDIES have shown a relationship between body weight and hypertension; weight gain increases the risk of hypertension developing. The mechanisms responsible for hypertension in obese patients remain obscure. Neither increased plasma volume, cardiac output, salt intake nor altered adrenergic mechanisms are adequate explanations alone. Obesity increases the cardiovascular risk; part of this increased risk can be ascribed to its association with hypertension. An additional risk occurs because of an increased cardiac output. Both hypertension and an increased stroke volume can contribute to the development of left ventricular hypertrophy and left ventricular failure. Weight loss in both moderately obese and morbidly obese persons will result in a significant reduction of blood pressure and the regression of left ventricular mass.

A lowered blood pressure occurs both in patients who have not received medication for hypertension and those who are already receiving medication. There is no linear relationship between weight loss and blood pressure reduction. It has been suggested that there is a "floor" in the blood pressure, so that the blood pressure will not reduce further despite continued weight loss. It is not clear at what level of weight reduction a floor in the blood pressure will be reached, but even a moderate weight reduction of 5 to 7 kg (10 to 15 lb) will produce a significant improvement in blood pressure control. This degree of weight loss will be more easily achieved than will a return to an ideal body weight. As many as 39% of hypertensive patients can remain normotensive for as long as four years without medication if a weight loss of 5 kg or more is coupled with reduced salt and alcohol intake.

The control of obesity is an important variable in managing patients with hypertension. Physicians should attempt to motivate their patients to lose weight if they are obese. If they succeed, better blood pressure control with fewer antihypertensive drugs should be the result.

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Relaxation Therapy for Hypertension

THE WORD "HYPERTENSION" to most patients implies a condition related to increased stress that by some mechanism results in an elevation of the arterial pressure. Although the role of stress as an etiologic factor in the development of hypertension remains a controversial issue, the effective application of relaxation therapy in managing hypertension is becoming clearer. In recent years nondrug therapies have been increasingly advocated in the initial management of mild hypertension, especially when the diastolic blood pressure remains in the range of 90 to 94 mm of mercury. Furthermore, nonpharmacologic approaches are a useful adjunct in treating moderate or severe hypertension and lead to the use of fewer medications in lower doses. Even though the morbid outcomes from untreated hypertension have not been shown to be lowered by the use of nonpharmacologic therapy, these interventions are generally accepted on the basis that they are not associated with significant risks or side effects.

There are more than 60 published studies on the use of transcendental meditation, yoga, several biofeedback techniques and teaching muscular relaxation in the therapy for hypertension. Most studies have shown a small but statistically significant reduction in systolic and diastolic blood pressures in the range of 2 to 10 mm of mercury, but several have shown only transient effects. The lack of carefully controlled studies using random assignment and blinded measurements and the absence of follow-up beyond eight months in most studies raise serious concerns about the effectiveness of these techniques in a clinical setting.

The first long-term follow-up study of relaxation therapy for mild hypertension was recently published in the *British Medical Journal*. In the original study, 204 patients with newly recognized untreated hypertension were randomly assigned to a relaxation program or a control group with fol-

low-up only. The intervention consisted of eight weekly one-hour group biofeedback sessions and prescribed muscular relaxation techniques twice a day at home. Of the 107 persons assigned to the experimental group, 99 accepted the treatment, and at eight months of follow-up they had a mean reduction in blood pressure of 16.2/6.8 mm of mercury compared with 5.0/0.3 mm of mercury for the control subjects. A four-year follow-up of 86 experimental and 75 control subjects found that the effectiveness of relaxation therapy was maintained at a reduced level. The subjects who had been taught relaxation therapy had a mean reduction of 7.2/3.7 mm of mercury compared with a mean increase in the control group of 1.6/4.1 mm of mercury. Although only 14 of the 86 participants practiced relaxation at least weekly, most (63%) practiced it occasionally.

The mechanism by which relaxation therapies lower blood pressure has not been extensively studied, but may be mediated by lowering of the plasma levels of catecholamines, renin and aldosterone. Patients' expectations may also have a significant role in the effect observed with relaxation training, and a subset may exist of hyperresponsive persons who are likely to benefit. In some of the published studies, less than half of the patients were willing to continue relaxation therapy for more than a few months, and thus it is likely that in clinical practice only a minority of patients having hypertension will actually benefit.

The Health and Public Policy Committee of the American College of Physicians recently concluded that biofeedback is a second-line nonpharmacologic therapy for hypertension. Relaxation training is cheaper, easier to implement and has a greater chance for a wider application in clinical practice. Nonphysician health professionals such as nutritionists and psychologists need to be involved in implementing the nonpharmacologic management of hypertension. Teaching relaxation therapy, including the use of commercially available audiotapes, can play an important role in a multicomponent non-drug approach to treating hypertension.

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Postprandial Hypotension

ANGINA PECTORIS AND SYNCOPE occur in some patients after large meals. In one study, a standardized breakfast for patients aged 75 to 98 years produced a mean fall in the systolic blood pressure—taken in the sitting position—of 13 mm of mercury within 35 minutes after ingesting the meal; a group of subjects aged 18 to 37 years had no significant reduction in blood pressure.

We have found that a meal produced a mean reduction in the supine diastolic blood pressure in normal subjects aged 19 to 31, associated with a reflexive increase in the mean heart rate. In patients with hypertension, a meal also reduced the mean supine diastolic blood pressure, associated with an increase in heart rate. Patients with autonomic dysfunction have a much more dramatic change in blood pressure after meals, averaging a decrease of 49 mm of mercury in the supine systolic blood pressure. Decreases as great as 98 mm of mercury have been seen.

The meal size appears to be an important determinant of hemodynamic effects, and meals with a higher protein content may have greater effects. Ingesting food increases the splanchnic blood flow and decreases total peripheral resistance, probably due to a decreased splanchnic resistance.

In many studies, elderly patients have been observed to have a greater hypotensive response to meals. In these studies, however, the blood pressure was higher in the older patients, and the magnitude of the reduction in blood pressure after the meal was also related to the premeal blood pressure.

Ingesting food lowers both systolic and diastolic blood pressures. Elderly patients, patients with hypertension and patients with autonomic insufficiency tend to have the greatest reductions in blood pressure. The hemodynamic effects of meals may contribute to postprandial syncope and to postprandial angina because of a reflex tachycardia and decreased coronary perfusion pressure. More commonly, blood pressure changes of 5 to 20 mm of mercury related to meals may contribute significantly to an apparent blood pressure lability and may seriously impair the interpretation of blood pressure control in patients with hypertension.

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'Unloading' Therapy for Heart Failure

VASODILATOR THERAPY can provide substantial benefits to some patients with congestive heart failure. The rationale is based on the physiologic principles of reducing ventricular afterload and preload (hence the term "unloading" therapy). This results in an increased cardiac output and decreased systemic and pulmonary venous pressures. Symptoms of hypoperfusion and congestion are ameliorated. The peripheral vascular tone (both arterial and venous) is increased in most symptomatic patients with congestive heart failure. An increased systemic vascular resistance and resistance to left ventricular ejection by arterial vasoconstriction reduces the forward stroke volume and cardiac output. Venoconstriction contributes to congestive symptoms by increasing pulmonary and systemic venous pressures; the intracardiac volume is also increased. The vasodilator drugs with predominantly arteriolar effects, such as hydralazine hydrochloride, minoxidil and nifedipine, increase cardiac output. Venodilators, such as nitroglycerin, nitrates and molsidomine, can decrease systemic and pulmonary venous pressures. Vasodilator agents with balanced arteriodilator and venodilator properties—angiotensin-converting enzyme inhibitors and α-adrenergic blocking agents—produce hemodynamic effects similar to those of combinations of arteriodilators and venodilators.

During congestive heart failure, abnormalities of neuroendocrine function contribute to a heightened peripheral vascular tone. Elevating levels of circulatory catecholamines reflect increased sympathetic activity. Plasma renin activity is increased in about two thirds of patients who have chronic